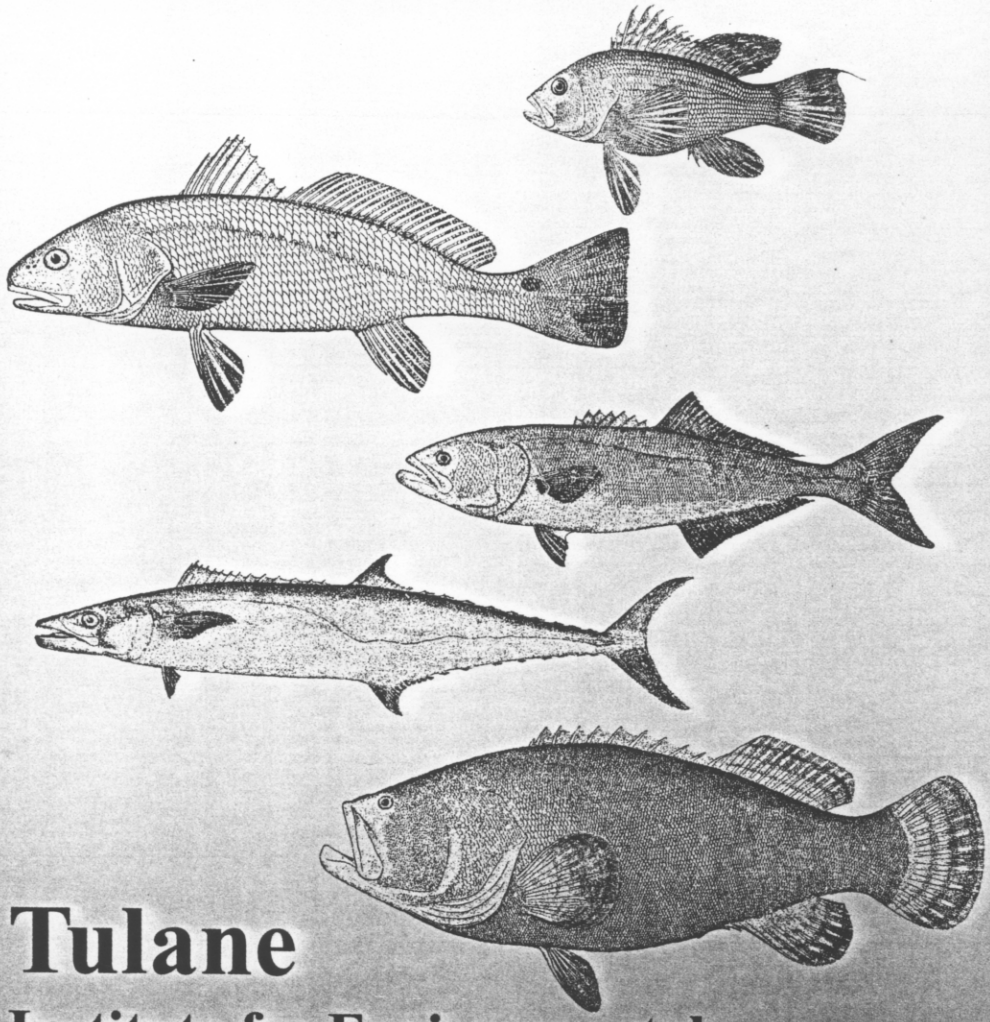
Sustainable Fisheries for the 21st Century?

A Critical Examination of Issues Associated with Implementing the Sustainable Fisheries Act



Tulane

Institute for Environmental Law and Policy

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HYPOXIC ZONES — The Looming Interface Between Land Pollution and the Sea

NATHALIE WALKER: Today we are going to talk about hypoxia or what is referred to as the "dead zone." In January of 1995, our organization (Earthjustice Legal Defense Fund) petitioned EPA, on behalf of eighteen environmental and fishing groups, to take some action to deal with the dead zone. That has lead to the creation of a federal task force on the issue and to the recent launch of a multi-disciplinary assessment group that is charged with probing the causes of and the potential solutions to the dead zone. We'll see. As I said, it was in 1995 that we asked EPA to take action, but we're patient people. We're happy that the infrastructure has been put in place, and we're very happy that folks like our speakers have been following the issue. We're going to start this morning with Dr. Nancy Rabalais. Dr. Rabalais is a professor at the Louisiana Universities Marine Consortium (LUMCON), where she has been for the last fourteen years. She teaches marine science courses at LUMCON and in the Department of Oceanography and Coastal Sciences at LSU. She has a Ph.D. in Zoology and a Bachelors and Masters in Biology. She has been studying hypoxic zones since 1985. She is going to be talking about the dead zone and fisheries impacts. The next speaker will also address fisheries impacts, and we'll end with a speaker who can give us a perspective on dead zones outside Louisiana.

NANCY RABALAIS: Welcome to the session on hypoxia in the Gulf of Mexico. What I will do is explain what hypoxia is, what causes it, and some of the implications for fisheries resources. Hypoxia is defined generally as low oxygen. The specific level we use to define it is anything less than two mg/l or ppm. The zone of hypoxia in the Gulf of Mexico is shown on this graph for mid-summer of 1993, '94, and '95. You can see that it extends from the Mississippi delta westward towards the Texas coast. In 1993, it extended a little into the Texas coast. It runs from very near shore to sixty kilometers off the coast, depending on the location along the coast. It is found in very shallow waters right near the beach out to about sixty meters. Its usual depth is between five and thirty meters. It is primarily a summer occurrence. It occurs most severely and is most wide-spread in June, July, and August, but it has been known to occur as early as February and as late as October. The 1996 data are not shown here, but the size was almost the same as 1995.

The 1997 zone was somewhat smaller, 15,840 square kilometers. We had a hurricane spawn in the middle of our study area and move throughout the southeastern coast in 1997, and that may have influenced the smaller size. The size of the zone doubled in 1993 from the average of the previous eight years. There was a record high flow for the Mississippi River in 1993, which delivered additional nutrients to the system throughout the spring and into the summer when the flow is usually low.

These figures show the distribution for 1985, 1986, 1987, partially in 1989 and 1990, when the zone was smaller. But, the pattern is consistent with down—plume or down—current effects of the Mississippi River delta and the Atchafalaya River delta. The river is important in the development and maintenance of hypoxia. The reason that we define hypoxia at two mg/l for our studies is that trawl data show that if the value falls below two mg/l, you normally don't catch any shrimp in trawls, and the same is true for finfish. Otherwise, there is quite a bit of variability in the catch data.

What causes hypoxia? This (slide) shows a cross-section of the water column of the southeastern coast and illustrates a typical summertime situation. The cross-section illustrates near-shore to off-shore, to about 30 meters water depth. The lower salinity waters are on the surface, the fresh water being delivered from the Mississippi and Atchafalaya Rivers. The higher salinity Gulf waters, 35 parts per thousand, are on the bottom. In summer, the winds are less, there is less mixing of the water column, and the water column is more stable. The result is a two layer water system that is maintained for long periods. The two layer system is present for most of the year because the Mississippi delivers so much fresh water. Hurricanes and cold fronts can break down the water column, mix up the two layer water system and alleviate the low oxygen problem. Also, the riverine fresh water delivers nutrients, nitrogen, phosphorus and silicate, which are very important and support the phytoplankton productivity and eventually the fisheries. When you have an overload of organic material, the cells either sink directly to the bottom or are eaten by plankton and incorporated in the fecal pellets, and the fecal pellets settle to the bottom. About half the material produced on the surface of the water gets to the bottom. Bacteria decompose the carbon and use up oxygen in the process of decomposition. Oxygen is utilized at a much faster rate than it can be resupplied from the surface, especially when the density gradient is

present. The result is areas at the bottom that are much less than two mg/l of oxygen, less than one sometimes, and very often anoxic. Hypoxia is not just at the bottom. It reaches well into the water column, and in recent years it reached within two to three meters of the surface.

If you stack these cross-sections up through time, you can see the temporal and spatial variability of hypoxia in 1992 and 1993. Hypoxia comes and goes in the spring. Once it sets up, it is extensive, widespread, and severe over large areas of the continental shelf. Thus, hypoxia is not just a mid-summer phenomenon, as is demonstrated by our shelf-wide maps. We see hypoxia over extended periods, in extensive areas.

The Mississippi River is a major player, as I mentioned, in both the physics and the biology of the system. Most of the fresh water that is delivered from the Mississippi flows west. The Atchafalaya captures one-third of the flow of the Mississippi, and most of that also flows west. Peak discharge is in the spring, and most of the nutrients are discharged then. Most of the primary production that fuels hypoxia occurs in the spring, so what happens to the river in the spring, in terms of the nutrient discharge, is very important to the development and continuation of hypoxia into the summertime.

What has happened to hypoxia over the years? First, the Mississippi River quality has changed considerably. What has not changed is the amount of water delivered by the Mississippi River. There has been a slight increase in discharge over recent decades, but it is not that much. What has happened is that the river's nitrogen and phosphate loads have doubled, and the silicate has been cut in half. We now have a peak in nitrate concentration during the spring that did not occur historically. We have more nitrate than we had historically—and even more during the spring, when the production in surface waters is very important. These changes are all closely related to the amount of nitrogen and phosphorus fertilizer applied in the watershed.

What has happened in response is that the continental shelf ecosystem has changed over the same time period. People always ask how long has hypoxia been around. Historical data do not go back far enough for us to say whether it has always been around. There are data from the early 1970s that shows that it occurred then. Our systematic surveys began in 1985. Since actual raw data do not exist, we have had to look to the sediments to determine how the system has actually changed. These sediments tell us that, yes, the ecosystem has changed.

There are indicators that surface water production has increased, and more carbon is getting to the bottom. There are also other indicators that oxygen stress is worse that it has been historically. Those changes are consistent with changes in nutrient flows to the river, which are consistent with changes in land-use practices in the watershed.

These (slides) are U.S. Geological Survey data that show that most of the nitrogen influx that enters the river is from the upper Mississippi River system. Most of the influx is the result of agricultural activities; a small amount is from atmospheric deposition and municipal waste. The distribution of fertilizer applications in the watershed shows where some of the areas of concern are. Why is this an issue in a conference on the Magnuson-Stevens Act? Well, hypoxia, because of its size, duration, and severity, does have the potential to affect fisheries. Fish can be killed directly or they can be forced out of their habitats. The amount of suitable habitats can be reduced considerably in size. When they are forced out, they can be preyed upon more easily. There is a consistent pattern of what happens when the oxygen drops from two down to zero. First of all, we very seldom see dead fish on the bottom. When you see dead fish on the bottom, it's the result of a low oxygen mass being forced onto shore, trapping the fish and killing them. This happens rarely, but it does occur and massive kills result when it does. When the oxygen level starts to fall below two, those organisms that normally can swim start to die off. Below 1.5, those larger organisms that are not as motile, succumb to the low oxygen level and are killed. Below about one, the benthos, or the organisms that live in the sediment, start to show stress. This slide shows brittle starfish and anemones that have come out of the sediments. This is not typical; normally they are buried in the sediment. This also shows that larger predators are not feeding in these areas; otherwise these organisms would not be there. Eventually, between one and 0.5, the organisms that normally live in the sediments come up and lie on the surface of the sediments. When the oxygen level in the sediments becomes low enough, organisms that live in the sediments die off. There is a fairly linear decline in the number of species, individuals, and biomass. When you get fairly close to zero, there is a massive amount of sulfur-oxidizing bacteria that carpets the bottom of the seabed.

What all this means to fisheries is not yet clear. As nutrients increase, fisheries yield increases. But, at the point where there is seasonal hypoxia, which has happened in the Gulf of Mexico, or

permanent, bottom-water anoxia, certain parts of the community begin to decline in abundance. Where we are in the Gulf of Mexico, no one is sure. Based on results from other areas of the world, these parts of the graph (declines) are why I think we should be paying attention to hypoxia.

NATHALIE WALKER: Thank you Nancy. Our next speaker is Roger Zimmerman. Today he is going to talk more about the fisheries impact of the dead zone. Roger is director of the National Marine Fisheries Service Laboratory in Galveston. He specializes in marine and estuarine ecology. He has a Bachelors and Masters in zoology and a Ph.D. in marine sciences.

ROGER ZIMMERMAN: There is good evidence that hypoxia in the Gulf of Mexico has affected commercial shrimp and finfish fisheries of Louisiana. Also, as Nancy Rabalais pointed out, there is good reason to believe that hypoxia has affected other demersal and benthic non-fishery species as well. The concern for fisheries is that hypoxic conditions in recent years have expanded downstream of the Mississippi River outlet in area, frequency, and duration. Monitoring by Rabalais and associates at LUMCON sufficiently demonstrates that the hypoxic zone now occupies a larger area, lasts longer, and is more persistent than in the past.

In 1996, the extent of the zone was historically larger than ever before. In perspective, its size of seven thousand square miles during 1996 was larger than the entire area of Chesapeake Bay. More significantly, hypoxia on the Louisiana shelf has roughly doubled in size from 3,500 square miles to about 7,000 square miles over the past decade. During the 1980s, the zone's area remained under 3,500 sq. miles. But during the 1990s, the zone increased in size, with a remarkably significant rise during 1993, after record-setting floods of the Mississippi River. Since then, the area of hypoxia has remained similarly large, leading to speculation that latent effects of the 1993 flood continue to maintain the zone's size into 1997. A pertinent question is how disruptive has this enlarged, prolonged environmental feature of the shelf landscape become.

The two most important commercial shrimp species in Louisiana, the brown shrimp, *Penaeus aztecus*, and the white shrimp, *Penaeus setiferus*, have life cycles that overlap in habitats with the

hypoxic zone both nearshore and offshore. For this reason, shrimp populations are likely to be highly susceptible to disruption by hypoxia. Brown shrimp and white shrimp spawn on the Louisiana shelf. Their larvae immigrate via currents into the estuaries of inshore coastal Louisiana. Resulting postlarvae metamorphose into small juvenile shrimp that grow within these estuarine nurseries. After about two months, large sub-adult juveniles emigrate from the nursery and return to the nearshore and offshore shelf to complete their growth into adults. The life cycle from egg to adult takes about 6 months. Larval, post-larval, sub-adult and adult shrimp move through and use pelagic and benthic habitats within the hypoxic zone. Depending on which stage within the life cycle, spawning grounds, feeding grounds and migratory pathways are impacted.

From a fisheries standpoint, hypoxia can be shown to directly influence the presence or absence of commercially important shrimp and finfish in the northwestern Gulf shelf ecosystem. The disruption can be demonstrated by comparing survey trawls taken inside and outside of the hypoxic zone. Plots of survey data from Leming and Stuntz published in 1984 demonstrate that, indeed, shrimp and finfish do not occur in hypoxic areas on the shelf. The highest biomass of shrimp and demersal fishes occur in areas with oxygen concentrations of 4 to 8 mg/l, whereas these fauna were virtually absent from trawls taken in waters with oxygen below 2 mg/l. Since shrimp and fish are mobile, and they were not reported to be found as dead carcasses in the survey trawls, it is presumed that such fauna move away from hypoxic conditions, perhaps with ensuing stress but little direct effect on mortality.

The principal effect of hypoxia is that mobile populations are re-distributed and concentrated into other, perhaps less preferred, areas or habitats. Other associated detrimental effects on bottom dwelling fishery species may include elimination or modification of feeding grounds and blockage of migratory or recruitment pathways. Each of the effects relate to important characteristics of Essential Fish Habitat (EFH), as defined in the Magnuson-Stevens Act. Therefore, we propose that, because it directly modifies and limits distribution of fishery species in normally used habitats of the Louisiana shelf, hypoxia is an EFH issue.

To test the hypothesis that shrimp fisheries are affected by hypoxia, we have analyzed commercial landings data in the northwestern Gulf of Mexico. Landings for the shrimp fishery are collected and reported by NMFS port agents located throughout the Gulf.

Monthly landings are reported by statistical sub-areas identified by geographic location and water depth. In short, the port agents collect landings data from dealers, interview captains for locations of catch, and thereby can determine catch and catch-per-unit-effort in hypoxic vs non-hypoxic zones off the coast of Louisiana. Since the landings databases are comprehensive and long-term, we can compare shrimp landings between geographic areas, depth zones and over time.

We have performed a cursory analysis for the Louisiana shelf including the area of hypoxia for the years between 1985 and 1995. Among geographical x depth statistical cells, we examined the relationship between size of shrimp catch and the percent of areal coverage of hypoxia within each cell. We found a significant negative relationship between shrimp catch and the percentage of hypoxia in cells using landings data from the months of July and August for years from 1985 through 1995. The relationship is quite definitive. In cells where hypoxia percent coverage (areal extent) is high, shrimp catches are always low. However, cells with no hypoxia demonstrate expected variability, i.e. both high and low shrimp catches .

Another parameter of shrimp fisheries is catch-per-unit-effort, referred to as CPUE. For CPUE, there is no relationship with percent coverage of hypoxia. This means that shrimp fishermen do not trawl where shrimp do not exist, nor where catch is too low to be economically viable. Normal CPUE in statistical cells with a high percent of hypoxia coverage strongly suggests that the presence of hypoxia is ephemeral. The implication is that hypoxia moves, comes and goes, emphasizing a dynamic nature. Shrimp are dynamic as well, and the population can easily move in and out of areas on the shelf. Whenever oxygen concentrations decrease, shrimp may detect, even anticipate, the presence of hypoxic conditions.

Laboratory experiments by Renaud (1986) show that brown shrimp and white shrimp detect low levels of oxygen and will move away from it to water with more oxygen. Further evidence that the shrimp actually may leave the area of low oxygen is apparent by examination of distribution of the shrimp catch in GIS maps for 1985, 1990 and 1994. By superimposing an outline of the hypoxic area onto a map with distribution of shrimp catch on the Louisiana coast, we see spatial relationships between shrimp catch and hypoxia. The gradient in shrimp catch from nearshore to offshore is notably steep. Shrimp catches diminish significantly coincident with the hypoxic zone.

Even more apparent is the lack of shrimp catch beyond the zone in offshore waters where sufficient oxygen exists. This appears to be due to hypoxia blocking the offshore migration of shrimp. When shrimp are blocked by hypoxia from moving offshore, the result is even lower catches offshore beyond hypoxia than within the hypoxic zone itself. Conversely, since migration offshore is restricted, the nearshore concentrations of shrimp remain high and catches are consistently large.

Furthermore, the presence of some catch within the hypoxic area indicates that hypoxia is not always present, that it is ephemeral. These patterns are similar year-after-year, and they are expected effects, i.e. (1) high catches nearshore, (2) reduced catch in the area impacted by hypoxia and (3) an absence of catch beyond the hypoxic zone. It is noteworthy that the scale of catch is orders of magnitude higher nearshore than offshore. The higher catch also coincides with higher effort directed to the nearshore fishery. As in other areas of the Gulf, the level of effort is stimulated by shrimp abundance. This unusually high level of nearshore effort justifies vigilance against overfishing, especially of adult white shrimp stocks, which spawn nearshore. In sum, there is evidence of an effect of hypoxia on fisheries and in particular for shrimp fisheries on the coastal shelf of Louisiana. It is evident that shrimp populations redistribute relative to hypoxic conditions. In part, shrimp avoid hypoxic waters and, as a result, their migration offshore has been blocked. Moreover, that large part of the shelf impacted by hypoxia to the extent of causing infaunal mortalities is removed or at least modified as feeding grounds for shrimp. Even when shrimp move back onto these feeding grounds after cessation of hypoxia, the community of annelid worms and other infaunal prey for shrimp is gone or reduced. No doubt, this reduction in prey can affect productivity of the shrimp population. Annual shrimp catches off Louisiana vary significantly and at least a component of downward variability may be due to large-scale hypoxic effects. Although the question is open on the extent to which the decline is due to hypoxia, reasonable concern is not.

NATHALIE WALKER: Thank you Roger. Now we are going to hear a perspective from someone who knows about a hypoxic area other than Louisiana. His name is Kent Mountford and he serves as the senior environmental scientist at the U.S. Environmental Protection Agency, Chesapeake Bay branch office. He's been there since its inception in 1984. He holds a Masters and Ph.D. in estuarine ecology and has been in

marine sciences for 33 years. He has lived and worked in the Chesapeake Bay area for 26 of those 33 years.

KENT MOUNTFORD: The Chesapeake Basin covers some 64,000 square miles. It stretches from New York to the North Carolina line. It is the largest estuary on this continent. Its a couple of hundred miles long, some thousands of square kilometers in area and relatively shallow in comparison to its proportions. I started working in the Chesapeake about this time of year 27 years ago. I was amazed to find that, like a big lake, it stratified each summer with warm, fresh water accumulating over the deep cooler bottom layer about May. This discontinuity we describe as a "pycnocline." Below this, as Nancy pointed out in the Gulf, accumulating organics sap much (if not all) of the oxygen, and, with no oxygen, all the higher life forms at least are extinguished. Through each summer, until autumn storms and falling temperatures break the cycle, its like a wave tank; the pycnocline can "rock" in response to weather events. In some cases, it rocks and sloshes low-oxygen water up on the shallows of Chesapeake Bay, affecting the organisms living on the bottom near the shore line. When that happens, you have "jubilees" in which any creature that can move is trying to get out of the water and away from the low oxygen.

Later work in this annually occurring dead zone allowed it to be measured and quantified. It has been tracked now for a couple of decades. We compute the volume of it from year to year and find that its about ten percent of the estuary volume. But below that density stratification of the pycnocline, about 30-40% of the Bay becomes uninhabitable for most life forms. So, it's a significant loss of habitat, live organisms, and therefore food supply in the estuary. The zone below low-oxygen water also becomes a chemical reactor, which makes it kind of a self-sustaining problem.

The root capability for this kind of thing goes back to the end of the Pleistocene era. The Bay was created by the flooding of the deep gorge that our Susquehanna River sawed into the coastal plain during our last Ice Age, which glaciologists call the Wisconsin Glaciation. This set our current Chesapeake Bay up with a deep axial channel from 90-174 feet deep as sea level rose and inundated coastal valleys. It used to be a coastal plain habitat. Some paleontologist friends of mine say that, after the Pleistocene, there were some periods of relatively severe ecological conditions caused by flows of unusual magnitude, organic materials from

forest fires. Such events are believed to have created hypoxic zones in that distant past. But my friends also say that in the period before and after European contact, say since the 1500s and 1600s, any prolonged anoxic event was unusual. It was not the norm. The frequency of these events, and their duration from the warm months sometimes well into October (as in the Gulf) are modern phenomena. They were just not experienced during the early years of colonization.

My agency, the EPA, funded research during the late 1970s and early 80s which indicated that hypoxia and anoxia had greatly increased during the last half century. The eventual linking of this information with data about nutrient enrichment from the watershed really "created" the Chesapeake Bay program.

The root cause of this problem, surprisingly, goes back to colonial agriculture and to Thomas Jefferson. Tom invented the moldboard plow. It turned over the soil rather than just tilling it, exposing the sod to erosive forces rather than just breaking the ground for planting. The result was a signal of soil erosion which appeared in the core samples taken deep in the sediments of the Bay, beginning about the middle of the 18th Century—about the time Tom and his plow got going, and erosion was immense thereafter. Add to this the first importation of fertilizers, which occurred about 1825-35 in the Chesapeake and, later, an exponential growth in chemical fertilizers, made possible by the conversion of WWII manufacturing capacity into agricultural capacity for the "green revolution"—and you see the problem.

This made the Bay not only a lot greener itself, but accelerated the depletion of oxygen as dying plankton fell to the Bay floor and decomposed. Agriculture does not bear the cross alone because, simultaneous with these trends, there was a tremendous explosion of conventional manufacturing, extractive industries, and the continuing post-WWII and Korean War population influx into the basin with its immense sewage discharges, loss of forest cover, increasing impervious surfaces and, of course, the still-swelling flood of vehicular traffic. Our Director, Bill Matuszeski says, "It's not people that are the problem, it's the lifestyles." People are not going to adopt seventeenth century lifestyles to reduce the environmental impact.

The problem, of course, is not just our activities themselves, but the nutrients that are released around the basin, nitrogen in particular. Generally, in estuaries, nitrogen is exhausted when plankton growth

starts peaking out and the plankton die; their celluar remains sink to the bottom, and their decomposition soaks up all the available oxygen. Water with no oxygen bathing the deep sediments becomes a chemical reactor, turning gray mud into a "black mayonnaise" from which re-mineralized nitrogen and phosphorus pour back out as dissolved nutrients into the water column and become "food" for the next generation of plankton "blooms." Its a self-sustaining wheel of life running on recycled nutrients that spins out of proportion to the annual load of fertilizers that are coming into the system from its rivers.

Why bother to clean it up? After all, might not the remineralization keep the system repeating itself for decades without any new contamination? Well, this isn't really likely. Researchers have projected that, if you get the bottom-dissolved oxygen up about one or two mg, you break the cycle of nutrient flux where remineralization occurs from the sediments. If you limit fertilization of plankton growth to that from nutrients arriving from the rivers, you will, thereafter pretty much reap what you sow.

Based on this premise, we went through a process of setting goals for dissolved oxygen. One goal is that we should not be below one mg/l at any time anywhere, and not below three mg/l for more than 12 hours in any 48-hour period. The monthly mean in the surface of the waters of the bay should also be five mg/l or greater, and any spawning area should always be above five mg/l. After studying the models, and ruminating on the political considerations, our Chesapeake Bay partner program people (the jurisdiction surrounding the Bay commission, which is the tri-state legislative commission, and the District of Columbia) all signed an agreement in 1987 to reduce controllable nutrient loads by 40% by the year 2000. This seemed far away, lots of time to reach the goal. People congratulated themselves, patted each other on the back and didn't do a whole lot. They are biting the bullet now.

The models project that when you achieve the goal of 40% reduction, you should get a change in bottom-dissolved oxygen significant enough to make meaningful improvements in Bay habitat. The implication is that abundant estuarine living resources will follow. We've made progress toward that goal.

With respect to phosphorus, we pretty much have the brass ring within reach. Nitrogen is a stickier case. It looks as if there could be a shortfall of 16 million pounds in the needed reduction of nitrogen for us to reach the 70 million pounds reduction goal. This is not all bad news.

We've achieved some massive reductions against an increasing tide of development in the basin. One of the problems of meeting the phosphorus goal but not the nitrogen is that, when you turn off the phosphorus, you tend to spill over excess nitrogen into the lower part of the system, where it can contribute to more algae growth. So you may achieve nutrient reduction upstream but get nutrient increases downstream. It is a tricky problem.

A whole slew of gap closures are under consideration, including efforts to recruit more agriculture volunteers to help with nutrient management programs, integrated pest management, and various manure management schemes designed to prevent nutrients from leaching into groundwater and percolating toward the Bay. This groundwater issue is complex, and there is evidence that fertilizers applied during WWII are just now reaching tributary water courses. Because of slow groundwater travel times, managers worry that good work done today may not produce substantial effects for a long time. Some considerable focus is being placed on sewage treatment plants, which are quicker fixes, though the retrofit of biological nutrient reduction technology, while effective, is not cheap. We pay for our past excesses.

A case in point: Because of a law suit against three Maryland counties and against my agency, EPA, we started working years ago to clean up the Patuxent River (the Chesapeake's sixth largest river) and got a jump start on both point and non-point discharge reductions. As of last year there was a relative reduction of 55-60% from the 1980s, in both nitrogen and phosphorus. As we reduced these levels over the last few years, the river has visibly begun to respond with higher oxygen levels, first upstream then downstream. I live in this watershed, and I am a volunteer water quality monitor in a program run by the Alliance for Chesapeake Bay. Among other things, I look at bottom-dissolved oxygen data every week or so, and I can report that the levels are better than in the 1970s, before the nutrient level peaked. So, I think these reductions are working. I was quite surprised to see this response, and I'm quite surprised to see it so soon. We'll see what happens over the next few years, Bay-wide. We've also had extremely high river flows three out of the last four years, and this of course increases the nutrient flows. About five years ago, we adopted public environmental indicators for the Bay. One of those indicators tracks the acreage of Bay grasses, which are one the Bay's most important habitats. The hope is that, Bay-wide, the increasing water transparency and the lower nutrient levels will

encourage the come back of this submerged aquatic vegetation, which hit a low during the 1980s. This is a bumpy road, but one worth traveling because it appears to be a real link between water quality, dissolved oxygen, and living resources.